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VERTIGO

ITS NEUROLOGICAL, OTOLOGICAL, CIRCULATORY, AND SURGICAL ASPECTS *

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The Nature of Vertigo

The word "vertigo" by its derivation implies a sensation of turning either of the body or of its surroundings, and it is so defined in the dictionaries. We need, however, a word to describe any subjective accompaniment of disordered equilibrium, whether this be a sense of rotation or not, and I propose, therefore, to use "vertigo" in this extended sense and to define it as "the consciousness of disordered orientation of the body in space."

There are three main ways in which the spatial orientation of the body may be felt to be disordered. (1) The external world, especially the world as seen, may appear to move. Such hallucination of movement is by no means always one of rotation. (2) The body itself may be felt to be moving, again not always in a rotatory fashion; or the sensation of movement may be referred to within the body—for example, within the head. (3) Finally, the postures and movements of the limbs, especially the lower limbs in standing and walking, are felt to be ill adjusted and unsteady.

To understand how vertigo arises it is necessary to begin with the physiological basis of the orientation of the body in space. Fundamentally this is reflex, and therefore unconscious. As is well known, an animal which has undergone a transection of the brain stem above the level of the red nuclei is capable of adopting a normal posture and of readjusting itself to this posture by means of righting reflexes if it is displaced from it. Such an animal, deprived of its cerebral hemispheres, is dependent upon the labyrinths and the proprioceptors of the muscles and joints of the limbs and spine for the stimuli which evoke from the centres of the brain stem and cerebellum a co-ordinated, and within limits appropriate, reflex response.

The role of the cerebral hemispheres in spatial orientation is to bring these reflex mechanisms into the service of the conscious perception of the external world. To achieve this the cerebral cortex must link together (1) afferent paths excited by stimuli from the exteroceptors, especially the retinae, which in the perception of visual space are closely associated with proprioceptor impulses from the ciliary and extra-ocular muscles; (2) proprioceptor impulses from all parts of the body which yield information as to the position of the body in space and of its parts relatively to one another; and (3) efferent paths through which voluntary movements modify the position of the body in space.

These are the two great corticifugal paths—the pyramidal tract, which links the precentral cortex directly with the anterior horn cells; and the fronto-pontine and temporo-pontine fibres, which bring the cortex into connexion with the cerebellum and so indirectly with the red nuclei, the postural reflex centres of the mid-brain.

THE PSYCHO-PHYSIOLOGICAL ASPECT

Vertigo, however, is a state of consciousness, and we must now consider it from a psycho-physiological point of view. In this task the methods of Gestalt psychology are helpful. There is a large field of conscious phenomena which are perceived as localized in space. Within this field we distinguish that portion of space occupied by our own body from the rest. The body is represented by a constantly changing model—a "plastic schema," to use Head's (1920) phrase. The external world is represented by another schema, also constantly being modified. These two schemata, however, are far from independent of each other, since the bodily schema receives contributions from proprioceptors, such as the labyrinths, which are in part stimulated by an external force, gravity, and, on the other hand, the perception of external space includes in its raw material proprioceptor impulses. The two schemata therefore to some extent interpenetrate one another, and must be conceived as functional and not as structural units. For consciousness the orientation of the body in space is normally an orderly dynamic relation between the bodily schema and the schema of the external world. Vertigo is the state of consciousness which arises when this relation becomes disordered.

Two illustrations may help to make this clear. As Hughlings Jackson (1932) pointed out, the simplest form of vertigo is that which is produced by paralysis of an ocular muscle. This is due to faulty projection of the

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visual field of the affected eye, as a result of which the patient is presented with two mutually conflicting visual spaces. The second illustration, which I owe to Hartmann (1935), is especially interesting in view of the important part which hallucinations of movement play in the symptomatology of vertigo. The perception of relative movement between the body and the external world must depend upon a certain functional relation between the bodily schema and the schema of the external world. It is not difficult to devise an experiment whereby a rotation of the visual field causes an hallucination that the body is moving in the opposite direction although the subject remains seated throughout the experiment in a chair. A familiar example of the same hallucination is the experience of a person seated in a train who, when a train beside his own begins to move, feels that his own train is moving in the opposite direction. It is clear from this and other observations that whether a perception of relative movement between the body and the external world is referred to the body or to the external world depends not only upon the content of visual perception but also upon the state of the bodily proprioceptors at the time, especially those of the ocular muscles and the labyrinths. If, owing to labyrinthine disease, a patient feels as if his body is in violent motion in a certain direction, and if nevertheless the objects at which he is looking remain in front of his eyes, he will experience an hallucination that the external world is moving in the direction in which he feels himself to move, for although he feels himself to be moving there is no cortical process corresponding to relative movement between his body and the external world. The latter, therefore, is "perceived" as moving in the same direction as the body.

It is worth noting, in passing, that the fact that a person can experience an hallucination of rotation of both his body and the external world in the same direction implies a space in reference to which they are perceived as rotating and which itself remains stationary. It appears, therefore, that our schemata of both the body and the external world are built up within an absolute space which is conditioned by our sensory organs and nervous system, and which forms a frame of reference within which the whole visible world, including the body, may be "perceived" as moving. This purely personal absolute space is one of the obstacles which the non-mathematical student encounters in trying to grasp Einstein's conception that all movement is relative.

Since vertigo is a state of consciousness in which spatial elements play an important part it is mediated by the cerebral cortex even when it is the outcome of a disorder at a lower level. It follows that conversely a disorder of the cortex may cause vertigo. Foerster (1936), in the course of electrical stimulation of the cerebral cortex in conscious patients, has observed that "stimulation of the superior lip of the interparietal sulcus evokes violent vertigo. The patient sees the objects before him moving towards the side of stimulation, or he has the sensation of turning towards the contralateral side, although objectively nothing happens and no turning of the head and the trunk really occurs." It is significant that at the cortical level stimulation of the same region may evoke an hallucination of rotation either of the body in one direction or of the environment in the opposite direction. This suggests that what is evoked primarily is that functional relation between the body schema and the schema of the external world which indicates relative movement between them. Which is felt to be moving and which is felt to be stationary probably depends upon the pre-existing background provided by the state of the proprioceptors of the body.

Vertigo as a Symptom of Disorder at Different Levels

It follows from the anatomical and physiological considerations already outlined that vertigo may arise as a result of a disturbance of function at many different levels. We therefore recognize (1) psychogenic vertigo, (2) vertigo due to cortical disturbances, (3) vertigo of ocular origin, (4) vertigo of cerebellar origin, (5) vertigo due to brain-stem lesions, (6) vertigo due to lesions of the eighth nerve, and (7) aural vertigo. In diffuse conditions, such as head injury and circulatory disease, it may be impossible to say whether the vertigo is cortical, brain-stem, or aural in origin.

(1) *Psychogenic Vertigo.*—"Giddiness" is a common complaint among sufferers from anxiety neurosis. There is no sensation of rotation, but the patient describes "a feeling as if I am going to fall," which usually occurs only when he is in the street, especially alone, and may be so severe that he is unable to go on or has to return home. A closely related neurotic symptom is "a feeling as if I am walking on air." Psychogenic vertigo is usually associated with severe feelings of anxiety and the symptoms of over-activity of the sympathetic nervous system. At the psycho-physiological level neurotic vertigo can perhaps best be explained as the effect of bringing into consciousness elements in spatial orientation (the relation of the body schema to the external world schema) which are normally unconscious, the cause of this being that this physical relationship has become for the patient symbolical of a psychological relationship between himself and his environment which is a source of difficulty to him. Such a process of symbolization is well illustrated by the double (physical and moral) meaning of the words "lapse," "fall," "fallen," and "downfall." Vertigo may also occur as a conversion symptom in hysteria.

(2) *Vertigo due to Cortical Disturbances.*—Foerster's observations, previously described, explain how an epileptic cortical discharge may cause a feeling of vertigo, as is not uncommon in petit mal, and how vertigo may arise as a symptom of migraine or of localized cortical lesions. In cases of intracranial tumour, however, vertigo is of no localizing value, since it may occur with a tumour in any situation (Symonds, 1933).

(3) *Vertigo of Ocular Origin.*—An explanation of vertigo associated with diplopia has already been given. Vertigo may also arise as a result of a difficulty in adapting the posture of the body to an unusual visual environment, such as a rapidly moving train seen from the railway platform. Vertigo on looking down from a height is a similar experience.

(4) *Vertigo of Cerebellar Origin.*—The role of the cerebellum in the causation of vertigo is difficult to assess, since many cerebellar lesions may involve also the vestibular fibres in the eighth nerve or the pons. Certainly vertigo may be slight or absent in spite of a massive lesion of the cerebellum, especially if this be limited to the lateral lobe. Nevertheless, most clinicians would probably agree that vertigo of cerebellar origin does occur, especially when the lesion involves the inferior vermis, which is closely linked anatomically with the vestibular system. This presumably is the cause of the vertigo which occurs at the onset of thrombosis of the posterior inferior cerebellar artery, and which is distinguished from

aural vertigo by the absence of deafness and tinnitus and the presence of the characteristic analgesia and thermo-anaesthesia of the face on the side of the lesion and of the body on the opposite side, due to the medullary infarction.

(5) *Vertigo due to Brain-stem Lesions.*—Vertigo may be caused by vascular or neoplastic lesions of the brain stem, but is most strikingly seen when disseminated sclerosis involves the pons. A young adult is suddenly attacked by severe vertigo accompanied by conspicuous nystagmus, and sometimes vomiting and prostration severe enough to cause confusion with aural vertigo. The absence of tinnitus and deafness, however, the presence of other signs of a pontine lesion, especially paresis of one external rectus, or of conjugate lateral gaze and facial paresis, together with other signs of disseminated sclerosis, and in some cases a history of previous nervous symptoms, enable the correct diagnosis to be made. In most cases, moreover, the vertigo as a rule lasts for several days, considerably longer than an attack of aural vertigo usually does.

(6) *Vertigo due to Lesions of the Eighth Nerve.*—Vertigo due to a lesion of the eighth nerve, especially acoustic neuroma, since it is associated with deafness and tinnitus closely simulates aural vertigo. Vertigo due to pressure upon the eighth nerve by abnormal vessels has been described by Holmes (1924) and Dandy (1937).

(7) Aural Vertigo

Vertigo is a symptom of a large number of pathological states of the ear, concerning which there are still many unsolved problems. I shall leave on one side the varieties of acute labyrinthitis, including infection of the internal ear secondary to suppurative otitis media, and I can only mention the interesting question of the role of the otoliths in vertigo. I shall confine myself to certain problems associated with Ménière's syndrome.

MÉNIÈRE'S SYNDROME

Recurrent aural vertigo arising in a previously healthy person is a clinical entity, though we cannot yet say whether it is also a pathological entity. The term "Ménière's syndrome" is used to-day less often than formerly, partly perhaps because a false idea of what Ménière described has been perpetuated by those who have not troubled to read his original communications (1860–1, 1861). It is said that Ménière reported an example of spontaneous haemorrhage into the labyrinth, a rare condition which clearly is not responsible for recurrent aural vertigo. It is true that Ménière did briefly report such a case to demonstrate that vertigo might be produced by a lesion of the labyrinth, but he also gave a clear and masterly account of all the essential clinical features of recurrent aural vertigo.

I would therefore urge the retention of the term "Ménière's syndrome" to describe the disorder characterized by the features to which Ménière originally drew attention—namely, (1) the sudden onset in (2) a previously healthy auditory apparatus of (3) functional disorders consisting of (4) continuous or intermittent tinnitus with (5) diminution of hearing and (6) vertigo, uncertain gait, rotations, and falling which are accompanied by (7) nausea, vomiting, and a syncopal state. Finally he noted (8) the progressive character of the deafness, and observed that the hearing might be suddenly and completely abolished.

There is little that the modern clinician can add to this classical description, except perhaps to mention (9) the transitory loss of vision which may occur without loss of consciousness during an attack (Gowers, 1907) and (10) transitory diplopia. It is important to remember that double vision may occur as a result of vestibular disorder, lest this symptom should be attributed to ophthalmoplegia and ascribed to a lesion of the nervous system. The two images are seen one above the other, and the diplopia is doubtless due to skew deviation of the eyes, a disorder of ocular posture emanating from the labyrinth and sometimes occurring transitorily, as Cairns and I have shown (1933), after section of the auditory nerve. It is also important to note that, as Ménière observed, loss of consciousness may occur in the course of a severe attack of aural vertigo, and such attacks require to be distinguished from epilepsy.

AETIOLOGY OF MÉNIÈRE'S SYNDROME

Men suffer from Ménière's syndrome more often than women in a proportion of about three to two. Age is an important factor. Ménière's syndrome is a disorder of middle age, especially late middle age. The age of onset of vertigo in a series of forty-one of my own patients is set out in the following table, the average being 49:

Age	Number	Percentage
10–19	1	2.4
20–29	2	4.8
30–39	6	14.6
40–49	9	22.0
50–59	8	19.5
60–69	13	31.7
70–79	2	5.0

This age incidence suggests the operation either of a degenerative factor or of some process requiring many years to cause symptoms.

There are two unusual types of patient, however, who do not accord well with the conception of a progressive disorder. Whereas the usual mode of onset is for tinnitus and deafness to precede vertigo by only a few months or at most a year or two, it occasionally happens that a patient who has been deaf in one ear for thirty or forty years suddenly develops vertigo. Here, perhaps, we are dealing with two different processes, one of which is superimposed upon the other. Even more puzzling are those unusual patients whose vertigo occurs at extremely long intervals. Thus a trained nurse aged 50 had a severe attack of giddiness and vomiting in 1918, another in 1932, another in 1937, and another, during which I saw her, in 1938. Occasional very mild attacks occurred in the intervals, but tinnitus and deafness were absent.

Focal aetiological factors include infection of the teeth, tonsils, and nasal sinuses, and I agree with Wright (1938a, 1938b) that focal sepsis is the most important single aetiological factor. I invite discussion as to whether the recurrent vertigo which is sometimes associated with blockage of the Eustachian canal is a pathologically distinct variety of aural vertigo.

Abnormalities of water metabolism have been emphasized by Mygind and Dederding (1928–9), Dederding (1929, 1931), and Nielsen (1931). Nielsen has found that sufferers from Ménière's syndrome tend to retain water in the body, while Furstenberg and his collaborators (1934) have precipitated attacks by giving chloride and so causing water retention, and both Dederding and Furstenberg have relieved them by dehydration. The affinity between recurrent aural vertigo and migraine was first pointed out by Ménière himself and has lately been

stressed by Dederding. There are certainly clinical resemblances, and the association of the two disorders in the same patient is not uncommon. Allergy may in some cases be a common basis (Balyeat, 1933).

The recent valuable observations of Cairns and Hallpike (1938), who have demonstrated a gross dilatation of the endolymph system in two cases, have made it easy to understand how changes in the osmotic tension of the blood reflected in the distended endolymph channels might cause variation in the symptoms and so explain the periodicity of the vertigo and also the fluctuations in the hearing noted by Dederding (1929). It seems unlikely, however, that disturbed water metabolism is the primary cause of the disorder, though it may well be a contributory factor. Here there are suggestive analogies with glaucoma. In the present state of our knowledge it seems best to regard Ménière's syndrome as the end-result of a toxic or toxi-infective process in the labyrinth —Wright's "focal labyrinthitis."

Treatment

If this view is right it follows that the eradication of any source of infection is the most radical form of treatment. Tobacco must be included among the exogenous poisons to which the labyrinth may be unusually susceptible in some individuals. When the intake of fluid can be shown to be excessive its restriction may be helpful, though few patients will tolerate the heroic measures sometimes recommended. Restriction to one and a half pints of fluid daily should not involve hardship. Striking improvement sometimes follows lumbar puncture, possibly as a result of its indirect effect upon water metabolism. Phenobarbital in ½-grain doses two or three times a day is most valuable. Cooper (1938) and Starr (1936) claim good results from certain choline compounds in a small series of patients. Most patients respond well to medical treatment if it is sufficiently thorough and comprehensive. Resistant and incapacitating vertigo, however, will rightly force the physician's hand, and surgical measures must then be contemplated. As in trigeminal neuralgia the choice lies between alcoholic injection and nerve division. Alcoholic injection of the labyrinth has its advocates (Mollison, 1936; Peacock, 1938; Wright, 1938b), but I have no personal experience of it. Surgical division of the eighth nerve limited to its vestibular fibres is practically free from risk and gives excellent results (Crowe, 1938).

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CO-OPERATION BETWEEN OPHTHALMOLOGIST AND PHYSICIAN IN CERTAIN CASES OF VISUAL LOSS*

BY

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Under our present system of medical practice the patients who come to an ophthalmologist are by no means all sent by their family doctor—a good many of them have not even seen him for years, and the first intimation he gets that all is not well with them is a letter from the specialist. Others who have seen their own doctor do not always take him fully into their confidence and do not realize that the condition of their eyes has any connexion with their health, and the family doctor himself does not always realize the connexion between the eye condition and the general condition of the patient.

It was with these ideas in my head that I suggested that a few remarks on collaboration between the ophthalmologist and the general practitioner, the physician and the neurologist, etc., might be of some value. As an example of what often happens I should like to read to you the letters which passed between me and one of my colleagues quite recently:

MY DEAR L.,—Mrs. A. came to see me yesterday complaining that her vision seemed to be slightly defective. She has worn glasses for some years and has a fair degree of hypermetropia and astigmatism, and the glasses did need some alteration. I could not get the vision of either eye up to full normal, the right one being slightly worse than the left. There is some slight pallor of both optic disks, especially the right, and, moreover, there is some rather vague limitation of the field of vision in the right eye. She says that her general health has not been very good, but I could not get any history which would connect up with early optic atrophy.

Have you had a Wassermann done? If it is positive, that would be a reason. The only other suggestion I can make is the possibility of a pituitary tumour, and I must confess that the fields of vision are not at all typical of that.

I am sorry to be so vague, but I feel that there is something wrong with the woman, but that is all I am certain of.

Yours sincerely,

C. B. F. TIVY.

DEAR TIVY,—Many thanks for your letter *re* Mrs. A. I believe you are seeing her again in a few weeks. You are quite right—there is something wrong. At your suggestion I got W—— to do a Wassermann and a Kahn test. Both are strongly positive, though she has not had any symptoms which would suggest this.

Going into her history, I find that her husband died some years ago of what appears to be G.P.I., and as she asked me point-blank if I thought she had had syphilis I saw no point in hedging.

I am starting treatment right away. She had her gall-stones, gall-bladder, and appendix out four years ago. I had her radiographed last year for digestive symptoms—a case of visceroptosis.

Yours sincerely,

L——.

* Read in opening a discussion in the Section of Ophthalmology at the Annual Meeting of the British Medical Association, Plymouth, 1938.